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FLUID FLOW IN THE LUNG: A SURVEY; NUMERICAL SOLUTION FOR GAS MIXING IN A SIMPLE MODEL OF THE LUNG; AND EXPERIMENTAL STUDY OF FLOW IN A THIN-WALLED COLLAPSIBLE TUBE

Jan David Janiec

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THESIS

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by

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June 1973

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Fluid Flow in the Lung: A Survey; Numerical Solution for Gas Mixing in a Simple Model of the Lung; and Experimental Study of Flow in a Thin-Walled Collapsible Tube

by

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Submitted in partial fulfillment of the
requirements for the degree of

MASTER OF SCIENCE IN AERONAUTICAL ENGINEERING

from the

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ABSTRACT

The range of fluid dynamics applications in the lung is vast, encompassing many interesting problems rarely touched in any other single engineering area. A survey of recent work in this field was conducted. Additionally, a numerical solution for gas mixing in a simple model of the lung was developed indicating its application to a wide range of similar problems. Finally, new parameters were experimentally investigated in flow through a thin-walled collapsible tube. Results indicated the effects of the parameters to be significant in the flow of blood in the pulmonary capillaries as controlled by the surrounding aveolar pressure.

TABLE OF CONTENTS

I. SURVEY OF FLUID DYNAMICS IN THE LUNG - - - - - - - 6

  A. INTRODUCTION - - - - - - - - - - - - - - - - - - - 6

  B. FLUID FLOW IN LARGE AIRWAYS AND ARTERIES - - - 6

  C. COMPLEX BRANCHING- - - - - - - - - - - - - - - 8

  D. BLOOD MICROCIRCULATION - - - - - - - - - - - - - 9

  E. FLUID PROPERTIES - - - - - - - - - - - - - - - - 11

  F. GAS MIXING IN THE RESPIRATORY TRACT- - - - - - 14

  G. GAS EXCHANGE AT THE ALVEOLAR INTERFACE - - - - 15

II. NUMERICAL SOLUTION FOR GAS MIXING IN A SIMPLE MODEL OF THE LUNG- - - - - - - - - - - 18

  A. INTRODUCTION - - - - - - - - - - - - - - - - - - 18

  B. THE SIMPLE MODEL - - - - - - - - - - - - - - - - 20

  C. ANALYSIS - 22

  D. SOLUTION - 24

  E. RESULTS AND DISCUSSION - - - - - - - - - - - - - 25

  F. CONCLUSIONS- - - - - - - - - - - - - - - - - - - 27

III. EXPERIMENTAL STUDY OF FLOW IN A THIN-WALLED COLLAPSIBLE TUBE- - - - - - - - - 28

  A. INTRODUCTION - - - - - - - - - - - - - - - - - - 28

  B. EXPERIMENTAL APPARATUS - - - - - - - - - - - - - 29

  C. EXPERIMENTAL PROCEDURE - - - - - - - - - - - - - 30

  D. RESULTS AND DISCUSSION - - - - - - - - - - - - - 31

  E. CONCLUSIONS- - - - - - - - - - - - - - - - - - - 32

IV. FIGURES- 33

APPENDIX A - NUMERICAL SOLUTION DATA - - - - - - - - - - - 48
APPENDIX B - THIN-WALLED COLLAPSIBLE TUBE DATA - - - - - 54
COMPUTER PROGRAM - 59
LIST OF REFERENCES - 61
BIBLIOGRAPHY - 63
INITIAL DISTRIBUTION LIST- - - - - - - - - - - - - - - - - 64
FORM DD 1473 - 65

LIST OF FIGURES

Figure No.	Title	Page
1	The Total System for Fluid Dynamics Applications	33
2	A Typical N_2 Washout Curve	34
3	One-dimensional Model of the Lung	34
4	N_2 Concentration during Inspiration, $T_{in} = 4.8$	35
5	N_2 Concentration during Inspiration, $N = 10$	36
6	N_2 Concentration during Expiration, $T_{in} = 12$, $N = 10$	37
7	N_2 Concentration during Expiration, $T_{in} = 48$, $N = 10$	38
8	N_2 Concentration during Expiration, $T_{in} = 96$, $N = 10$	39
9	N_2 Washout Curve, $T_{in} = 12$	40
10	N_2 Washout Curve, $T_{in} = 48$	41
11	N_2 Washout Curve, $T_{in} = 96$	42
12	Experimental Apparatus	43
13	Experimental Apparatus	43
14	Flow Variation with Alveolar Pressure, L/D = 5.5	44
15	Flow Variation with Alveolar Pressure, L/D = 4.0	44
16	Flow Variation with Alveolar Pressure, L/D = 2.5	45
17	"Parallel" Flow in Collapsible Tube	45
18	"Balloon" Flow in Collapsible Tube	46
19	Striations Prior to Collapse	46
20	Unsteady Collapse	47

I. SURVEY OF FLUID DYNAMICS IN THE LUNG

A. INTRODUCTION

The study of fluid dynamics in the lung is a wide field, encompassing a large range of problems. The vastness of the field is easily understood when the complexity of the organization of the respiratory tract and the cardiovascular system is considered. Many of the problems which arise in these areas are unique even when the fluid dynamics of the problems are considered in isolation. Often, these problems require work in areas of fluid dynamics that have never been attempted in purely engineering applications. Frequently, new researches in the field center on very basic fluid flow problems, and the results are as interesting as any that are suggested by engineering applications. Figure 1 indicates the main areas of study within the cardiovascular-respiratory system.

B. FLUID FLOW IN LARGE AIRWAYS AND ARTERIES

In both the large airways of the lung and in the larger arteries the Reynolds number may vary from several thousand to one or two hundred. This range of flows has not been closely studied, particularly the large Reynolds number end. In most engineering applications large Reynolds number studies have been limited to fully developed turbulent flow. In the lung, however, high Reynolds number flows are only sporadically or locally turbulent, seldom becoming fully developed.

In studies by Kuchar and Ostrach [Ref. 1] for large Reynolds number flow regimes including the effect of viscosity, pulsatile flow, and vessel elasticity, the developing flow was found to have a strong boundary layer character, resulting in high wall shear stresses near the vessel entrances. The development length was found to be large and dependent on the Reynolds number and unsteadiness parameter, while wall distensibility was not significant. Thus flow development characteristics are significant in the large airways and arteries.

Additionally, the large vessels may be subject to curvature, inducing large secondary flows due to centrifugal forces. These forces, acting as a result of curvature of the primary flow streamlines, produce secondary flow velocities at right angles to the primary streamlines. Study by McConologue and Srivastava [Ref. 2] has indicated that the resulting motion of a central fluid is toward the outside of the bend, and a return flow toward the inside of the bend near the wall. The motion toward the outside wall is nearly uniform while the return flow, retarded by viscous stress, is limited to a thin boundary layer. A result of this fact was the increase in critical Reynolds number based on pipe diameter for transition in curved pipes compared with straight pipes. In addition, the secondary flow promotes a much more rapid lateral mixing than would be possible for normal laminar flow.

Work has been undertaken to study the combined effect of the last two topics due to the fact that the first entry region to both the cardiovascular system and respiratory tract is a curved tube: the great arch of the Aorta, and the mouth and trachea.

C. COMPLEX BRANCHING

Complex branching of the passages exists in both the respiratory tract and the cardiovascular system. As the flow branches early in each system it undergoes a large distortion at each branch, and the flow may not recover before reaching the next bifurcation. In the smaller airways and capillaries where the Reynolds number is too small for this effect, the flow becomes difficult to analyze due to the large number of tubes in parallel and the lack of information on the distribution of tubes by length and diameter.

Pedley, Schroter, and Sudlow [Ref. 3] found that in the higher Reynolds number airways secondary flow was exhibited downstream of each bifurcation, with peak axial velocities just outside the boundary layer on the inner wall of the junction. These secondary motions decayed slowly downstream.

Additional study by Caro, Fitz-Gerald, and Schroter [Ref. 4] has indicated that the rate of shear in a branching system was dependent on two factors: a flow factor depending on the total flow in a particular tube, and a geometrical

factor. The geometrical factor was found to decrease downstream. It was also greater on the outside of a bend than on the inside. At a bifurcation the factor was largest on the central flow divider, the outer bends of the two daughter branches becoming inside bends, effectively. The flow factor was also found to decrease with distance downstream. The general result was that the rate of shear was least nearer the heart or mouth and, within these, at the site where the geometrical factor was smallest.

In steady flow a branching system affects pressure drops at each series of bifurcations. Here the effect of resistance differs for the arterial problem and the bronchial problem, as discussed by Lighthill [Ref. 5]. In the bloodstream the greatest pressure drops occur in the smaller vessels of diameter less than 0.1 mm., whereas in the airways the maximum pressure drops occur early in the branching, soon after the fifth generation. The airways maintain a much larger daughter-to-mother branch ratio closer to the system entrance. This coupled with the higher Reynolds number in those regions would tend to cause separation, a possible explanation of the phenomenon. The arterial resistance problem is said to be a low Reynolds number problem while bronchial resistance is considered a high Reynolds number problem.

D. BLOOD MICROCIRCULATION

Flow in the small capillaries of the lung differs greatly from that usually found in engineering fluid

dynamics. The feature which distinguishes this type of flow is that the fluid inertial effects are totally negligible. The Reynolds number is so low that the flow pattern is determined solely by the need for pressure gradients and viscous forces to be in exact equilibrium. Under these conditions, Bernoulli's equation is not applicable and there is essentially no difference between static and dynamic pressures. Centrifugal forces are also negligible: fluid can negotiate sharp bends with no secondary flow effects. In general, fluid motion is insensitive to vessel geometry and there is little tendency for separation. This insensitivity to geometry was further demonstrated by Fitz-Gerald [Ref. 6] with respect to entrance length effects. The difference between Poiseuille motion of a fluid and fluid entering a tube at a large angle, with the same total flow, was reduced by an order of magnitude in an axial distance of half the tube radius.

Propagation of changes in pressure, in the large vessels governed mainly by a balance between distensibility and inertia, is governed in the smaller vessels entirely by a balance between distensibility and resistivity. A feature of the blood circulation in most parts of the body is the vasomotor control of peripheral perfusion, the ability of smooth muscle to alter the diameter of small vessels in order to vary the flow rate through the vessels by an order of magnitude. Contrary to the systemic microcirculation system, microcirculation in the lung exhibits almost no

vasomotor control in the pre-capillary vessels or elsewhere. As noted by Lighthill [Ref. 5], most of the resistance to flow appears to be in the capillaries themselves.

Perfusion in the capillaries can vary greatly with location in the lung, an effect based predominantly on gravity as presented by Glaister [Ref. 7]. A typical pulmonary capillary is easily distorted by pressure differences between the capillary and alveolus. In effect, the capillary is a thin-walled collapsible tube in which flow is governed by the alveolar pressure P_a, and the upstream and downstream capillary pressures, P_1 and P_2, respectively. The following information was obtained and verified by Conrad [Ref. 8], Holt [Ref. 9], and many others. The effect of gravity was to divide the lung into three basic zones. In zone 1, at the top of the lung, P_a was greater than P_1 or P_2, and there was no flow. In zone 2, the middle of the lung, P_2 was less than P_a which was less than P_1, and flow was governed by the pressure difference $p_1 - p_a$. In zone 3, low in the lung, P_a was less than P_1 or P_2, and flow was unobstructed and proportional to the pressure difference $P_1 - P_2$. An experimental investigation of flow through a thin-walled collapsible tube is included in this paper in Section III.

E. FLUID PROPERTIES

A problem relatively unique to physiological fluid flow lies in the unusual fluid properties of blood, and to a lesser extent of air. Blood is a suspension (40-50% by

volume) of small deformable bodies, mainly red cells, in a transparent fluid, the blood plasma. Although the flow properties of plasma are similar to those described by Newton's viscosity law, the concentration of red cells at low strain rates yields substantially increasingly effective viscosities. However, Lighthill [Ref. 5] indicates that data suggest that at the higher strain rates in the larger vessels the errors are not large in treating the blood as a Newtonian fluid with constant viscosity. Studies of particulate suspensions by Mason and Goldsmith [Ref. 10], simulating blood over a range of Reynolds numbers extending from creeping viscous flow to that where inertial effects play an important role, indicated that at low Reynolds numbers for a single particle, particle deformation and interaction with the wall forced migration to the axis of the tube. At higher Reynolds numbers, particles also migrated laterally, but to an eccentric radial position. In blood-similar suspensions at low Reynolds numbers, blunting of the velocity profiles was evident, the dispersions were non-Newtonian, and cells were deformed significantly. At higher Reynolds numbers in steady flow, and in oscillatory and pulsatile flow, cells tended to migrate inward from the tube wall. This resulted in a two-phase flow of a central, more viscous core surrounded by a thin, particle-depleted and less viscous zone, with an attendant decrease in the energy dissipated in the tube.

Applications of "hydrodynamic lubrication theory" by Lighthill [Ref. 11] to blood flow in the narrowest capillaries have corresponded to the observed facts that the stress necessary to deform a capillary wall was nearly two orders of magnitude larger than that required to deform a red cell. Thus it is that the cells deform very substantially in order to pass through the smallest capillaries. Lubrication theory further suggests that the stress, which the wall of a very narrow capillary passage must transmit to a red cell sliding past it so that the cell will deform sufficiently, must be transmitted by viscous action in a thin lubricating layer of plasma; that the plasma film thickness must adjust passively to the value required; and that sliding may cease when the plasma film thickness becomes too small. Theoretical observations also indicated that the film thickness should vary with the square root of the cell velocity. This is an interesting non-linear effect.

Air is also a suspension of dust and other fine particles, and significant study has been devoted to the behavior of airborne particles in the lung. The deviation of particle motion from gas motion is caused by Brownian motion, gravity, and inertial forces. Altshuler [Ref. 12] stated that for particles of less than 0.2 μm the dominant force is Brownian motion. For particles of greater than 1 μm the dominant effects are settling and inertial displacement.

Owen [Ref. 13] observed that for inspired flow rates of 40 L./min. and greater the boundary layer on the tracheal

wall was largely turbulent. In addition, if the air entering the trachea separates at the vocal chords, almost fully developed turbulent pipe flow may exist and decay slowly. This would suggest that many particles are deposited on the tracheal walls; however, the normal rate of evaporation from the tracheal wall causes a radial motion sufficient to impede the deposition of all but the finest or coarsest particles.

G. GAS MIXING IN THE RESPIRATORY TRACT

At ordinary breathing frequencies the main mechanical impedance to gas flow within the lungs stems from the elastic distensibility of the air spaces terminating the airways (alveoli), the flow resistance of the air passages being extremely small. Due to the weight of the lungs, there is a gradient of increasing distension from bottom to top. Mead [Ref. 14] found that the lower, less distended regions were more compliant than the upper, more distended ones, and that there was a gradient of increasing gas flow from top to bottom. As a result, the upper regions of the lungs received only half the bulk flow of gas per gram of lung tissue received by the lower regions.

Davidson and Fitz-Gerald [Ref. 15] discovered that for various geometrical models for small airways in the lung, the bulk flow was significant for gas transport in the final generations of the lung.

In single-breath nitrogen washout experiments, as discussed by Farhi [Ref. 16] and Pedley [Ref. 17], in which

the subject inspired a single breath of pure oxygen, then expired it, the N_2 concentration was measured at the mouth during expiration. The results indicate that initially there is no N_2 because the expired gas is the pure O_2 most recently inspired, which never made contact with the N_2 rich air already present in the lung before O_2 inspiration began. Following this region, the N_2 concentration rapidly increased to a nearly constant value on the "alveolar plateau." The alveolar plateau represented air from deep in the lungs where the N_2 concentration was initially that of ordinary air.

Gas mixing in a simple model of the lung was solved numerically in Section II of this paper.

G. GAS EXCHANGE AT THE ALVEOLAR INTERFACE

The interchange of materials (O_2, CO_2) by the circulatory system depends on two processes: (1) circulation of blood through the capillary beds and (2) transport of various components of the blood through the capillary walls. Net transport across capillary walls is driven by two basic forces: (1) differences in hydrostatic or effective osmotic pressure of the fluids on either side (ultrafiltration transport) and (2) differences in concentrations of permeating solutes on either side (diffusion transport).

Renkin [Ref. 18] found that control of ultrafiltration transport was brought about by changes in the ratio of the resistance of the latter half of a capillary bed to the

total resistance of the bed, and by changes in the number of open capillaries. Control of diffusion transport was brought about by changes in the number of open capillaries, and by changes in local distribution of capillary blood flow relative to capillary surface and permeability.

As blood flows along the pulmonary capillaries it receives oxygen from the alveoli, and delivers carbon dioxide to them. Thus concentration gradients for the gases develop along the capillaries, increasing for oxygen and decreasing for carbon dioxide. Study of digital computer models of pulmonary gas exchange by Milhorn and Pulley [Ref. 19] revealed that the partial pressure of blood reached equilibrium with alveolar oxygen at about two-fifths of the total distance along the capillary pathway. Also the partial pressure of the CO_2 reached equilibrium with the blood about halfway along the capillary pathway. Additionally, the partial-pressure gradient of oxygen had a concave shape due to the convex shape of its dissociation curve. The concavity did not exist in either the concentration gradient of oxygen or in each of the curves for carbon dioxide.

The effect of pulsatile flow of blood on oxygenation of the blood in pulmonary capillaries for an ideal lung was investigated by Graeser, Kim, and Crandall [Ref. 20] for several combinations of exercise and atmospheric oxygen content. The pulsatile flow was found to give substantially the same end capillary alveolar-arterial tension gradient as that found for a constant velocity model. When breathing

air, the pulsatile flow effect yielded a 10-15% greater diffusing capacity than the constant velocity model, but no effect was observed in low oxygen cases.

II. NUMERICAL SOLUTION FOR GAS MIXING IN A SIMPLE MODEL OF THE LUNG

A. INTRODUCTION

The diffusion of gas throughout the lung is a complex problem dependent on both convective transfer and molecular diffusion. A typical physiological experiment in this field is the single breath nitrogen washout experiment described by Farhi [Ref. 16] and Pedley [Ref. 17]. The subject, starting from an arbitrary lung volume, inspired a single breath of pure oxygen. The subject then exhaled and the N_2 concentration at the mouth was measured during expiration. The resulting graph of N_2 concentration versus time was typically similar to that of Figure 2, in which high-frequency oscillations due to the cardiac cycle have been averaged out. In region I there was essentially no N_2 due to the fact that the expired gas was the pure O_2 most recently inspired, which never reached the N_2 rich air already in the lung upon inspiration. Region II was a transition region where the N_2 concentration increased rapidly with time, reaching a nearly constant level called the "alveolar plateau" in region III. The alveolar plateau represented air from deep in the lungs where the initial N_2 concentration was that of ordinary air. If the total volume of O_2 inspired (V_{in}) (related to time by the mass flow rate) was less than the anatomical dead space (V_D) (the volume of the airways as opposed to the alveoli), then

the volume V_0 at which the plateau was reached was approximately equal to V_{in}. If V_{in} was greater than V_D, V_0 was approximately equal to V_D, although the exact relationship was a function of the gas mixing and diffusion of the inspired gas with the air previously present.

A significant effect in region III was that the N_2 concentration was not constant, but exhibited a marked slope. There are two possible explanations for this phenomenon. The first is based on "regional inhomogeneity," that is, the concentration of N_2 in the alveoli near the top of the lung is less than at the bottom because the upper regions are initially more expanded than the lower due to the difference in pleural pressure. Thus, less oxygen may enter the lower regions during inspiration. This also requires that the air from the upper regions is expired first, often called "sequential emptying." There is evidence supporting regional inhomogeneity; however, sequential emptying is quite controversial. The second explanation is that of "stratified inhomogeneity," that is, there is a concentration gradient between the inspired O_2 and the N_2 rich gas already present, and there is no a priori reason to assume this gradient will not exist in the alveoli.

The simple model which follows was developed to study the gas mixing problem, and the N_2 washout case in particular.

B. THE SIMPLE MODEL

The basic development of this model was suggested by Pedley [Ref. 17].

It has been noted that the lung is a very complicated network of branched tubes, in which the exact velocity distribution remains unknown. Reynolds numbers may vary from several thousand to less than one. Thus, solution of the full diffusion equation, for which the fluid velocity field is required, is impossible, and considerable simplification is necessary.

The lung was assumed to be a symmetric, dichotomously branching system, in which all pathways have the same length. The lung was replaced with a one-dimensional system whose cross-sectional area $a(x)$ increased rapidly from the trachea to alveoli as shown in Figure 3 and consistent with Weibel's [Ref. 21] data. The area was considered constant from the mouth to the first branching of the airways, where it increased greatly. The cross-sectional area of the trachea, a_0, is approximately 2.5 cm^2. The area of the final generation of airways is of the order of 10^4 cm^2. The position where the area began to increase was taken to be $x = 0$. The distance from the mouth to $x = 0$ is about 38 cm, while the remaining distance to the final generation is about 6.6 cm. $a(x)$ was assumed to be a continuous smooth function independent of time. All volume change was considered to take place in the alveoli.

The N_2 concentration was considered to be uniform across a cross section. Thus, the average concentration c(x,t) was a function only of distance x down the lung and of time t. The velocity u(x,t) was also assumed to be uniform across a cross section. These assumptions were not as drastic as they first appear. The trumpet-shaped tube in Figure 3 is actually a large number of small tubes nearly in parallel, especially at the alveolar end. The main effect of radial velocities, velocity profiles, and turbulence is to influence the relationship of concentration to the longitudinal velocity. Compensation for this omission was introduced in the diffusivity.

Previous theories by Cuming, Crank, Horsfield, and Parker [Ref. 22] and La Force and Lewis [Ref. 23] have used similar models, but greatly oversimplified the actual gas mixing process. They ignored the fact that mixing takes place everywhere simultaneously with convection, especially in the upper airways. They assumed a period of convection, in which the interface between pure O_2 and mixed air remained flat, followed by a period of diffusion, followed by another period of convection. Secondly, they assumed that the diffusion was governed at all times by the molecular diffusivity D_{mol}. In reality, in both laminar and turbulent flow, the existence of a velocity profile or turbulent eddy ensures that longitudinal mixing is much greater than can be described by pure molecular diffusivity. Taylor [Refs. 24 and 25] found that after a certain time the average concentration could be

calculated from a knowledge of the average longitudinal velocity, on the basis of an effective longitudinal diffusivity much greater than D_{mol}.

Flow in the trachea was assumed to be turbulent and an effective longitudinal diffusivity (D_0) was calculated using Taylor's relation. For a tracheal Reynolds number of 5000, D_0 was approximately 260 cm^2/sec. In the terminal regions of the lung, the mixing is governed almost entirely by the molecular diffusivity with a value of about 0.2 cm^2/sec. In between these values, the flow undergoes large changes, about which little is known specifically. D(x) was considered to vary smoothly between D_0 and D_{mol} from $x = 0$ to the alveolar end. Therefore, the important feature of greatest mixing at the highest velocities was introduced.

C. ANALYSIS

The governing equation for diffusion in the one-dimensional model was:

$$\frac{\partial c}{\partial t} + u \frac{\partial c}{\partial x} = \frac{\partial}{\partial x} \left(D \frac{\partial c}{\partial x}\right) + \frac{D}{a} \frac{\partial a}{\partial x} \frac{\partial c}{\partial x}$$

where the last term accounts for the change in cross sectional area.

From continuity:

$$u(x,t) = \frac{u_0 a_0}{a(x)}$$

where u_0 was the velocity in the trachea, and a_0 was the cross-sectional area of the trachea. Inspiratory and expiratory flow rates were considered uniform and to be equal and opposite.

The boundary conditions on $c(x,t)$ were as follows. Both c and $\partial c/\partial x$ were continuous at $x = 0$. At the alveolar end, for N_2 there was no flux across the end, $\partial c/\partial x = 0$. At the mouth, $c = 0$ for inspiration (pure O_2) and $\partial c/\partial x = 0$ for expiration (diffusion negligible compared with convection).

The initial condition for inspiration was uniform concentration everywhere. The initial condition for expiration was the distribution of concentration at the end of inspiration.

In the governing equation for diffusion, the concentration was non-dimensionalized with respect to its initial value, and all other quantities with respect to their values in the trachea ($C = \frac{c}{c_0}$, $\bar{D} = \frac{D}{D_0}$, $X = \frac{x}{\ell}$, $A = \frac{a}{a_0}$, $T = \frac{tu_0}{\ell}$) .

The following equation was obtained:

$$\frac{\partial C}{\partial T} AN + [\pm N - \frac{\partial}{\partial X} (A\bar{D})] \frac{\partial C}{\partial X} = A\bar{D} \frac{\partial^2 C}{\partial X^2}$$

where the minus sign denotes inspiration, the plus sign for expiration, and $N = u_0\ell/D_0$, the effective Peclet number in the trachea.

The new boundary and initial conditions were:

$$C(X,0) = 1$$

for inspiration:

$$C(-5,T) = 0$$

$$\frac{\partial C}{\partial X}(1-\delta,T) = 0$$

for expiration:

$$\frac{\partial C}{\partial X}(-5,T) = \frac{\partial C}{\partial X}(1-\delta,T) = 0$$

where $\delta = (a_{alv}/a_0)^{-2}$, and ensured that for reasonable Peclet numbers, the values of A and $\bar{D}$ were close to their actual values ($\delta \simeq 0.02$), at $X = 1-\delta$.

The particular forms chosen for A(X) and $\bar{D}$(X) were:

$-5 \leq X \leq 0$ $\quad A = \bar{D} = 1$

$0 \leq X \leq (1-\delta)$ $\quad A = (1-X)^{-2}$

$0 \leq X \leq (1-\delta)$ $\quad \bar{D} = D_{mol} + \frac{1-D_{mol}}{A^2}$

The function of $A = (1-X)^{-2}$ was consistent with Weibel's data.

D. SOLUTION

Solution of the non-dimensional equation was performed numerically with the use of finite difference approximations: $\partial C/\partial T$ by forward difference, and $\partial C/\partial X$, $\partial^2 C/\partial X^2$ by central difference. The resulting equation was solved on the digital computer using sufficiently small time and distance steps to maintain stability and accuracy. The concentration at any

time T was calculated from data for the previous time step. Solutions were generated for four values of the effective Peclet number in the trachea. Inspiration time before expiration was also varied.

E. RESULTS AND DISCUSSION

The simplified model of the lung produced results similar to those described by Farhi [Ref. 16] and Pedley [Ref. 17], with a minor variation. In addition, several interesting results were observed.

The effective Peclet number in the trachea was found to have an effect that varied with distance down the model, as shown in Figure 4 and by data in Appendix A. In the trachea, an increase in the Peclet number caused a decrease in the N_2 concentration at a particular position. This effect was reversed for all X greater than zero, in the actual branching. That is, an increase in Peclet number increased the N_2 concentration at a particular position. In addition, this effect tended to be felt just upstream of X = 0. The general result was that as the Peclet number decreased, the further down the lung the diffusion progressed. This indicates that a reduced Peclet number corresponded to relative increase in the diffusivity with respect to the velocity.

The effect of the duration of inspiration was also dependent on distance down the model. In the trachea, the concentration rapidly decreased with time, while in the lung the process was much slower, as shown in Figure 5.

N_2 concentration in the model during expiration also varied considerably from the trachea to the lung. As expiration time increased, the N_2 concentration slowly increased linearly along the trachea, then rapidly increased with distance to the alveoli (Figures 6, 7, 8). Increasing expiration time also produced an increase in the N_2 concentration at a particular position, with the exception of the last few airways before the alveoli.

The N_2 washout curves obtained were similar to those expected with the exception that the sharper changes in slope were not detectable between regions II and III, as shown in Figures 9, 10, and 11. In fact, for the longest inspiration (T = 96), the curve for Peclet number of 12 was essentially linear. For this reason it was very difficult to examine the relationships between V_0, V_D, and V_{in} as discussed in the introduction. V_D was 55 (non-dimensional), and for Figure 9, $V_0 = 12$, and the relationship appeared to hold. For Figure 10, $V_{in} = V_0 = 48$, but it was difficult to determine exactly where the alveolar plateau began. For Figure 11, $V_0 = 96$ and the alveolar plateau should have begun at $V_D = 55$. It obviously did not for either case.

The effect of Peclet number was to decrease the concentration at a particular time with an increase in Peclet number for the washout curves.

F. CONCLUSIONS

The results of the numerical solution for the theoretical simple model of gas mixing in the lung justified the following conclusions:

1. The model effectively simulated gas mixing in the lung with simultaneous diffusion and convection effects.

2. The model is very flexible and many parameters may be easily varied to more closely approximate the experimental data for the washout curves.

3. The flexibility of the model extends to many other similar experiments in the field. The ease with which initial conditions, boundary conditions, and the various parameters may be varied and the rapidity of computer solution provide a fast, accurate tool for future research in the field.

III. EXPERIMENTAL STUDY OF FLOW IN A THIN-WALLED COLLAPSIBLE TUBE

A. INTRODUCTION

It was noted earlier, in the survey section of this paper, that flow in the small capillaries of the lung is analogous to flow in a thin-walled collapsible tube. Flow in this type of tube is dependent on the surrounding pressure as well as the upstream and downstream pressures.

The force of gravity exerted on the lung tends to divide it into three basic zones. In zone I at the top of the lung, the alveolar pressure P_A is greater than the upstream static pressure P_1. In zone II, near the middle of the lung, P_1 is greater than P_A which is in turn greater than the downstream static pressure P_2. In the lowest part of the lung, zone III, P_A is less than both P_1 and P_2. The pressure-flow relationships in these three zones have been experimentally studied by Conrad [Ref. 8] and Holt [Ref. 9] and many others. The results have all indicated that the flow is pressure dependent as follows. In zone I the tube was collapsed and there was no flow. In zone II the flow was proportional to $P_1 - P_A$, and the tube was observed to neck near the downstream end. In zone III the flow was unobstructed and proportional to $P_1 - P_2$. These results were obtained using a latex rubber tube as a model surrounded by an airtight box. The fluid involved was water. The results were based on a single length-to-

diameter (L/D) ratio and a single tube tension in each study.

This experiment was conducted using air as the fluid in order to verify the previous results and to study the effects of variations in L/D ratio and tension in the tube wall.

B. EXPERIMENTAL APPARATUS

The experimental apparatus used in this investigation was designed and constructed similar to the previous models of Conrad [Ref. 8] and Holt [Ref. 9]. Alterations were introduced where necessary to simplify the apparatus and adapt it to air flow. The system is shown in Figures 12 and 13. The details of the equipment were as follows.

An airtight box was constructed with dimensions 14" by 10" by 10" using 1/2" Plexiglas sheet. The inlet to the box was a decreasing radius nozzle constructed from powdered aluminum-filled epoxy resin. It converged smoothly from an outside diameter of 5-1/2" to an inner diameter of 1" aluminum pipe. The outside diameter of the pipe was 1-1/4". The exit from the box was via the same type pipe. The mouths of the two pipes were separated by 10", and radiused to ensure smooth flow. Variations in the L/D ratio were attained using several lengths of Plexiglas tubing adjusted over the aluminum pipe. The inside diameter of the Plexiglas tubing was 1-1/4", and the outside diameter was 1-1/2". The mouth of each tube was also radiused. A 1"

gate valve was mounted between the exit and the vacuum source in order to adjust the pressure. Pressure in the box was regulated by a 3/4" bleed from the exit pipe (taken upstream of the 1" gate valve) equipped with a 3/4" gate valve. Static pressure ports were located at the inlet, exit, and top of the box. A pitot tube was inserted in the exit flow. The pressure ports were connected to an inclined water manometer. The suction source was a Singer vacuum cleaner, Model D-5. The collapsible tube was a thin rubber prophylactic with a limp length and diameter of 7" and 1-3/8", respectively. It was stretched over the Plexiglas tube at each end, and fixed by rubber O-rings and bands.

C. EXPERIMENTAL PROCEDURE

The basic experimental procedure consisted of variation of tube tension for each of three L/D ratios.

The 1" gate valve was opened to adjust the inlet pressure P_1 to a value of 2.0 in H_2O below atmospheric pressure, and maintained at this level for all investigations. One of three L/D ratios was then chosen and the tube tension was adjusted for each of three values. A new L/D ratio was chosen and the procedure was repeated. The pressure in the box P_A was varied from 5.0 in H_2O below atmospheric up to collapse of the tube. As a tube was stretched, its diameter decreased, and the tension of the tube was quantified by the pressure difference $P_1 - P_A$ necessary to maintain

parallel flow in the tube (tube walls parallel to the pipe walls at the same diameter).

D. RESULTS AND DISCUSSION

The experimental study of air flow in a collapsible tube produced significant results. The data collected are tabulated in Appendix B and graphically shown in Figures 14, 15, and 16. In general, the results obtained were similar to those of previous work with water, and additional new information was observed.

In zone I the flow was discovered to differ from zero, depending on the initial tension of the tube. In the low tension case, flow was found to exist for P_A greater than P_1 before collapse of the tube. Generally, the effect of decreasing tension was to increase the pressure P_A attained before collapse. In zone II the slope of the dynamic pressure curve (directly related to the mass flow) rapidly increased as P_A passed P_2, and leveled as P_A approached (and passed in some cases) P_1. In zone III the major deviation from previous results occurred. As the difference $P_1 - P_2$ was maintained as a constant in each case, the dynamic pressure should have remained constant for all P_A less than P_1. The results of this investigation indicated that the flow was constant until parallel flow was attained. However, further decrease in P_A caused the cross section to balloon, accompanied by a drop in the dynamic pressure.

As depicted in Figures 14, 15, and 16, the effect of varying L/D ratios was to increase the P_A possible before collapse, with decreasing L/D.

Three qualitative phenomena were observed during experimentation. First, just prior to collapse, the tube walls developed longitudinal striations near the center of the tube. Second, collapse was a highly non-steady phenomenon. It was not complete, steady collapse, but a rapid oscillatory motion of the tube walls from complete collapse to the striated state described above. Finally, the collapse was observed to take place at a position approximately 1/5 of the tube length from the downstream end for each value of L/D. The "ballooning" effect was also observed to begin at the downstream end.

E. CONCLUSIONS

The results of the investigation of air flow in a thin-walled collapsible tube justified the following conclusions:

1. The experimental apparatus used was an accurate model of flow in a collapsible tube utilizing new parameter variations.

2. The length-to-diameter ratio of the flexible tube was found to have a significant effect on pressure-flow relationships.

3. The longitudinal tension of the flexible tube was also found to have a very significant effect on the pressure-flow relationships.

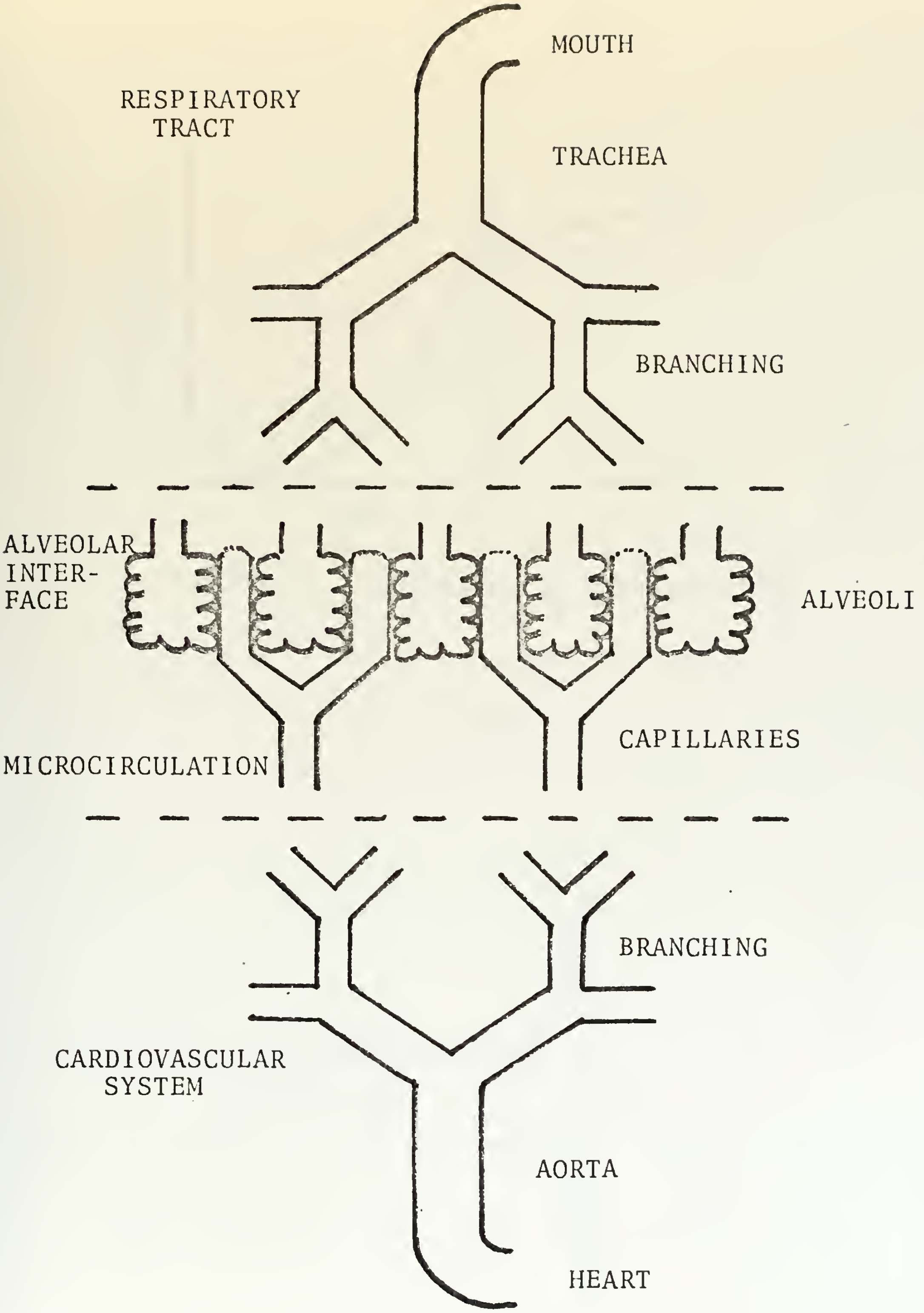

FIGURE 1. THE TOTAL SYSTEM FOR FLUID DYNAMICS APPLICATIONS.

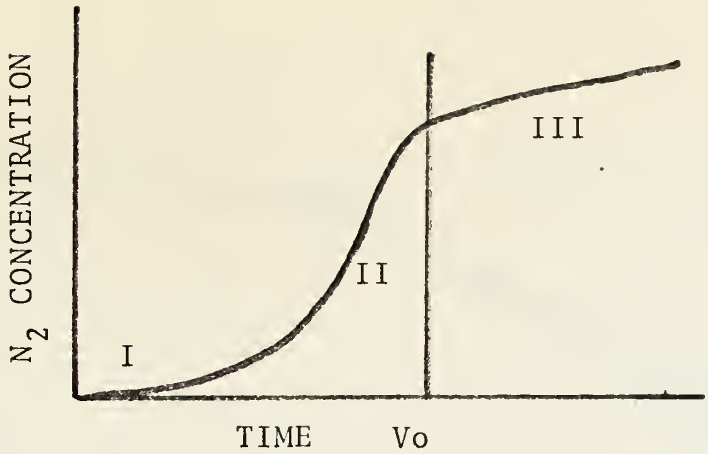

FIGURE 2. A TYPICAL N_2 WASHOUT CURVE

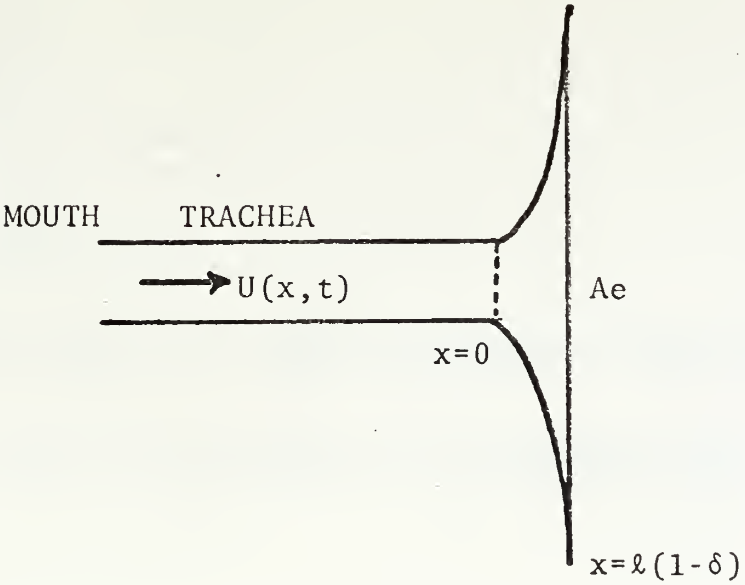

FIGURE 3. ONE-DIMENSIONAL MODEL OF THE LUNG

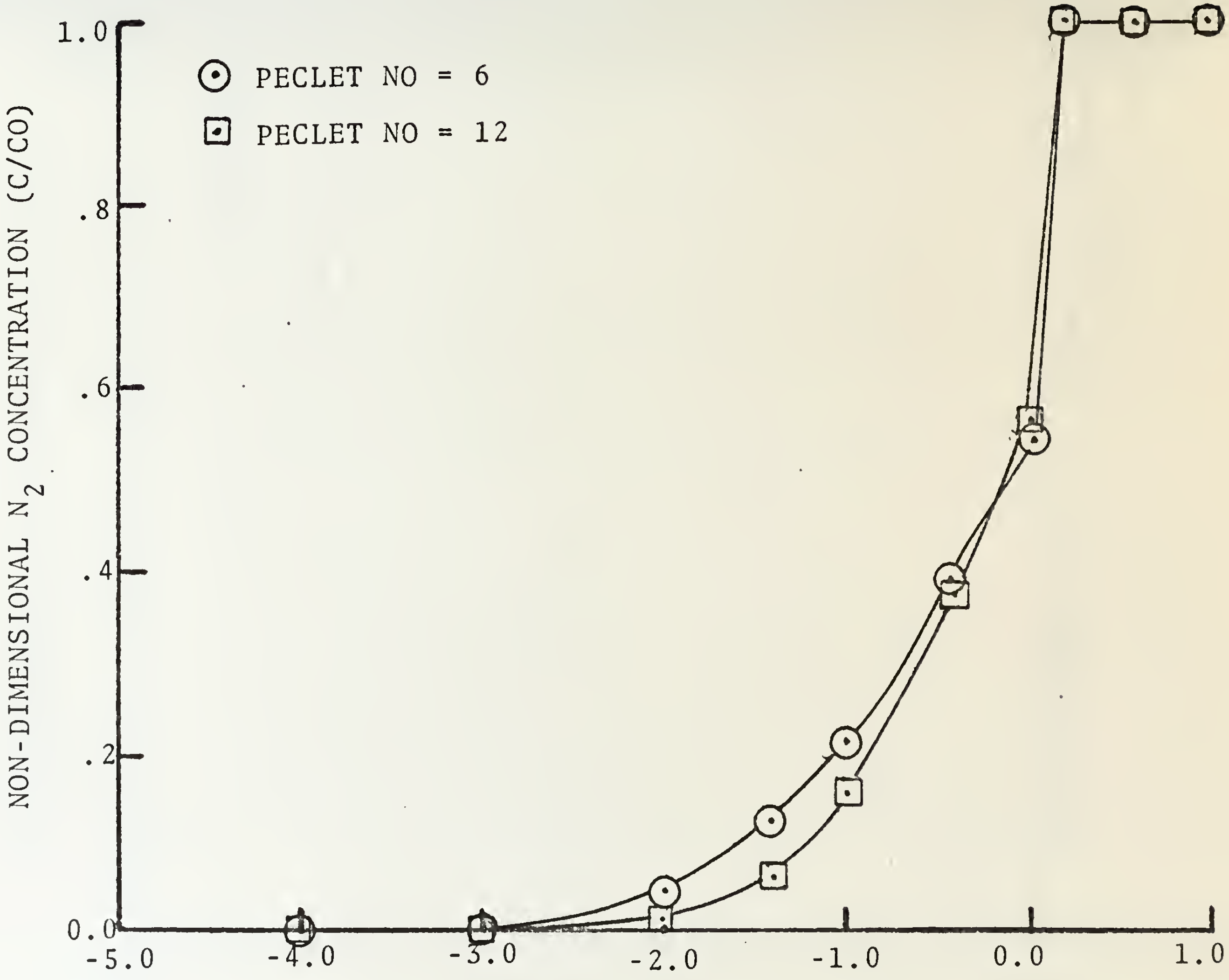

FIGURE 4. N_2 CONCENTRATION DURING INSPIRATION, T_{in} = 4.8.

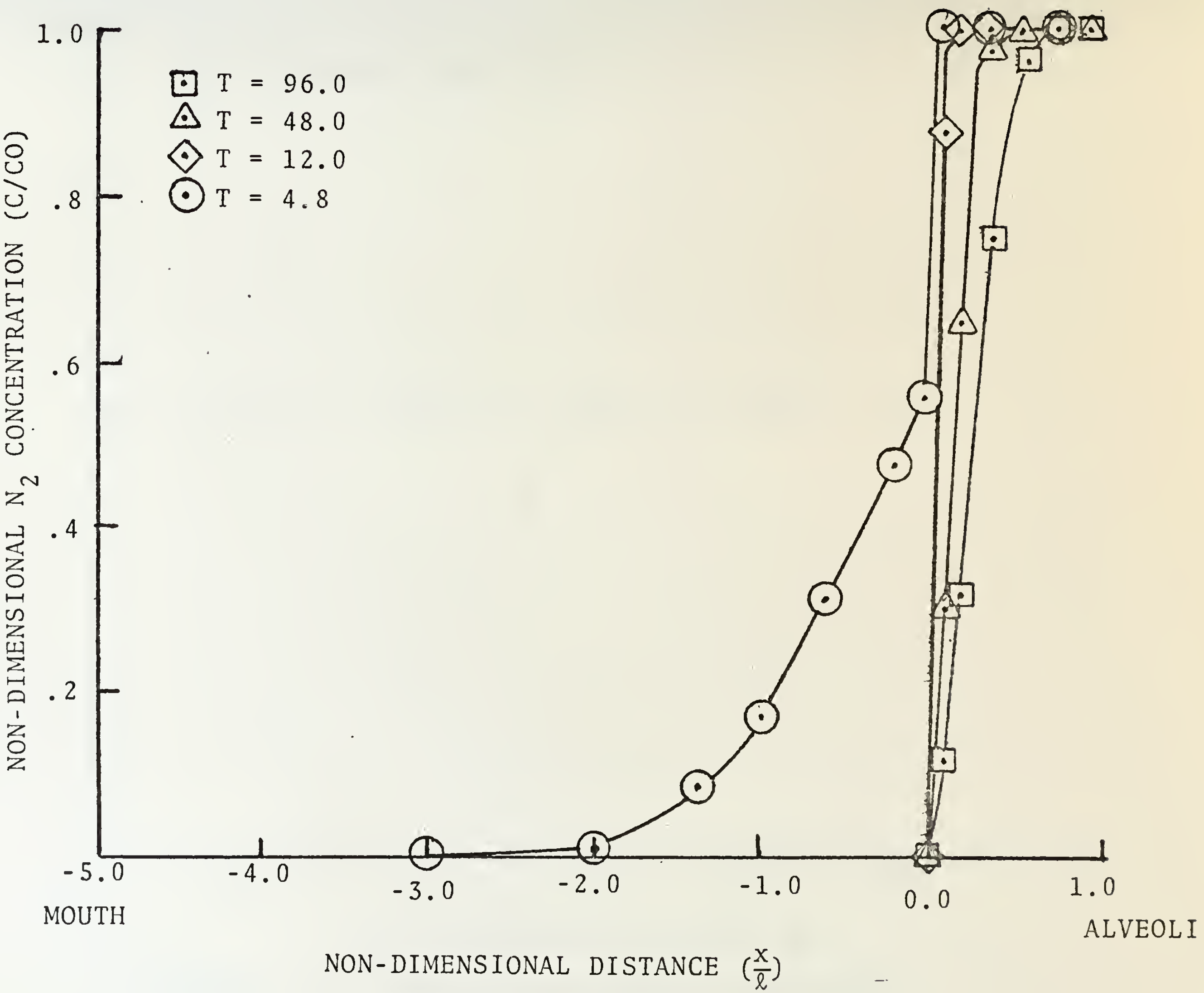

FIGURE 5. N_2 CONCENTRATION DURING INSPIRATION, N = 10.

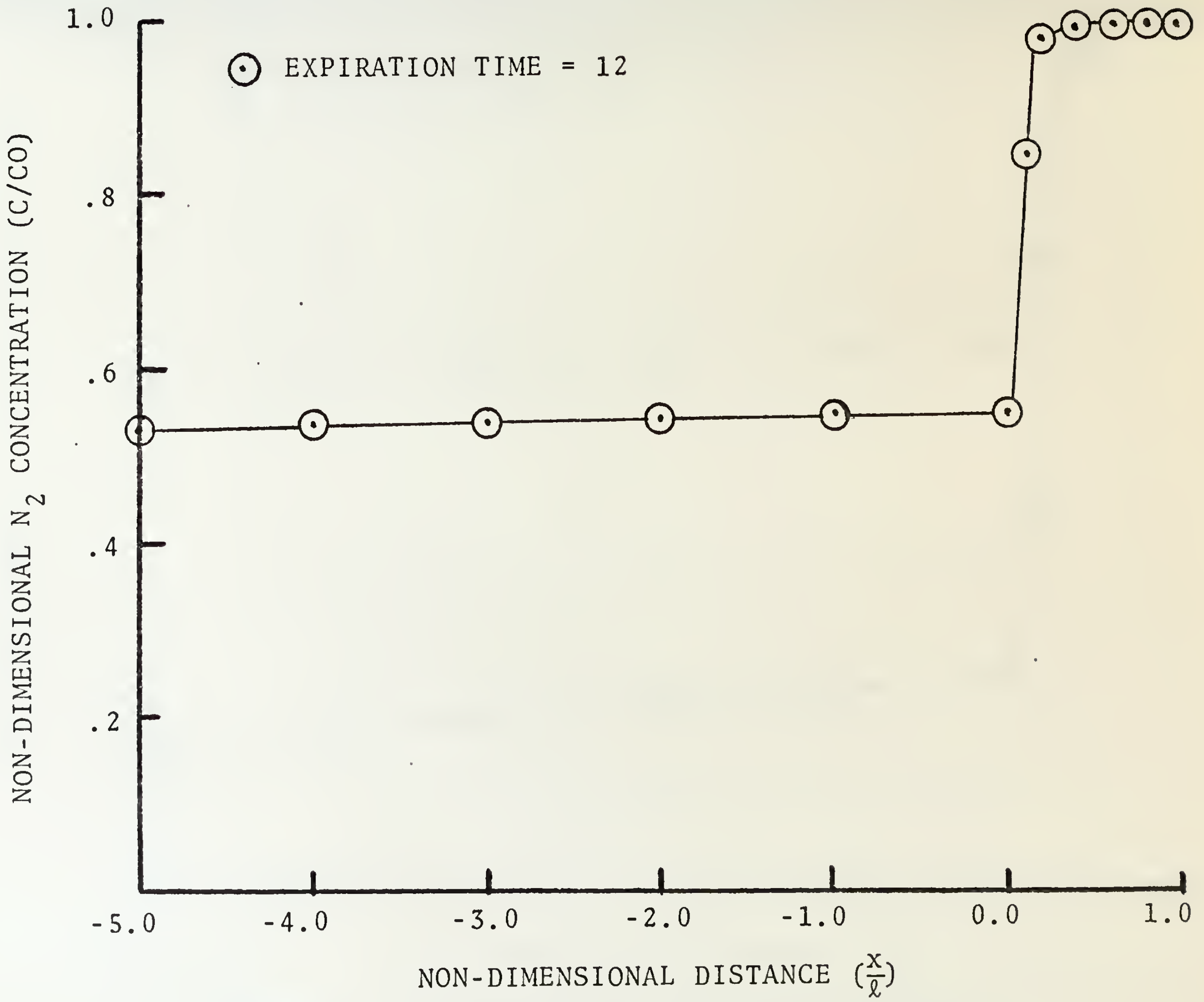

FIGURE 6. N_2 CONCENTRATION DURING EXPIRATION
$T_{in} = 12$, $N = 10$

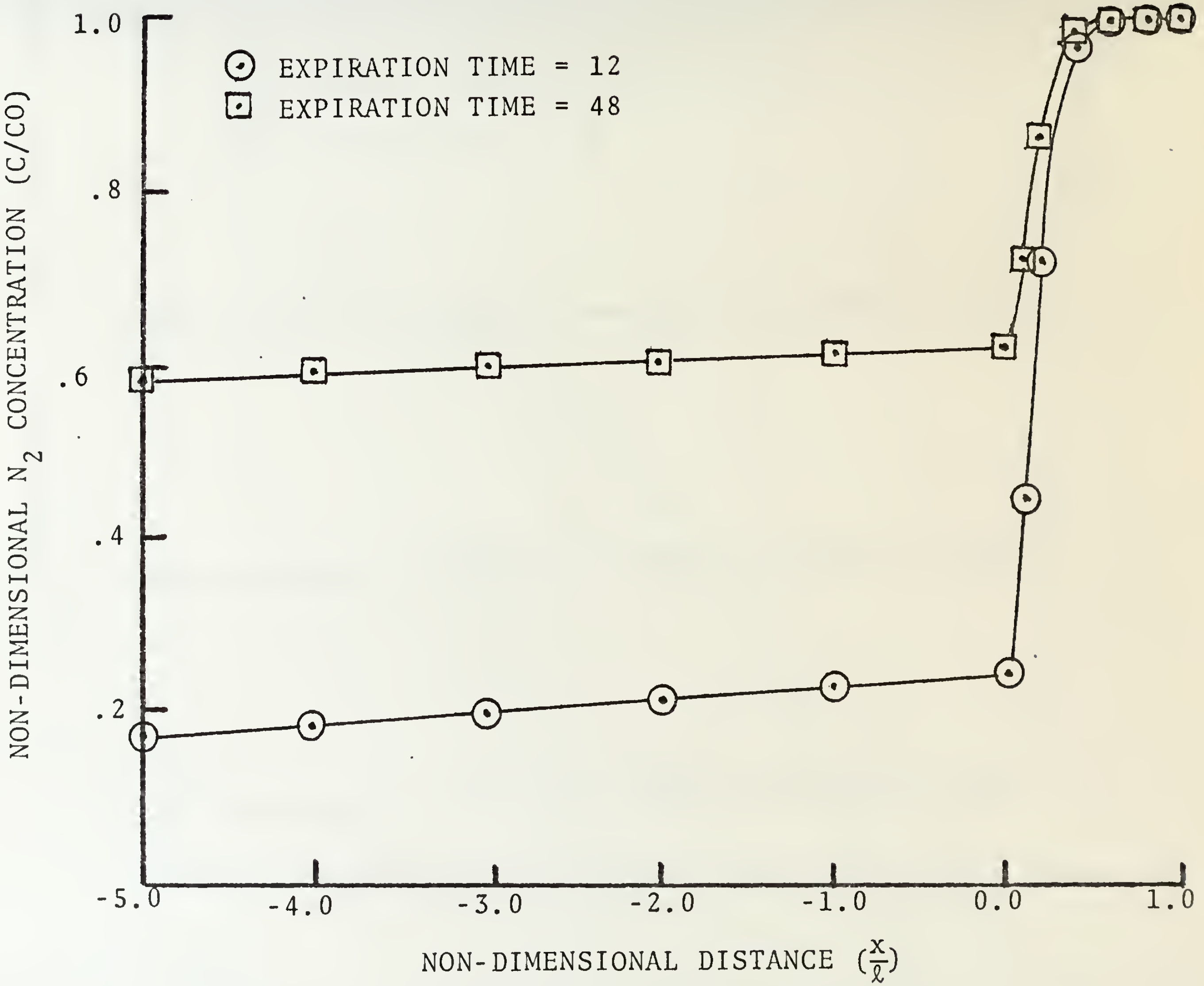

FIGURE 7. N_2 CONCENTRATION DURING EXPIRATION
$N = 10$, $T_{in} = 48$

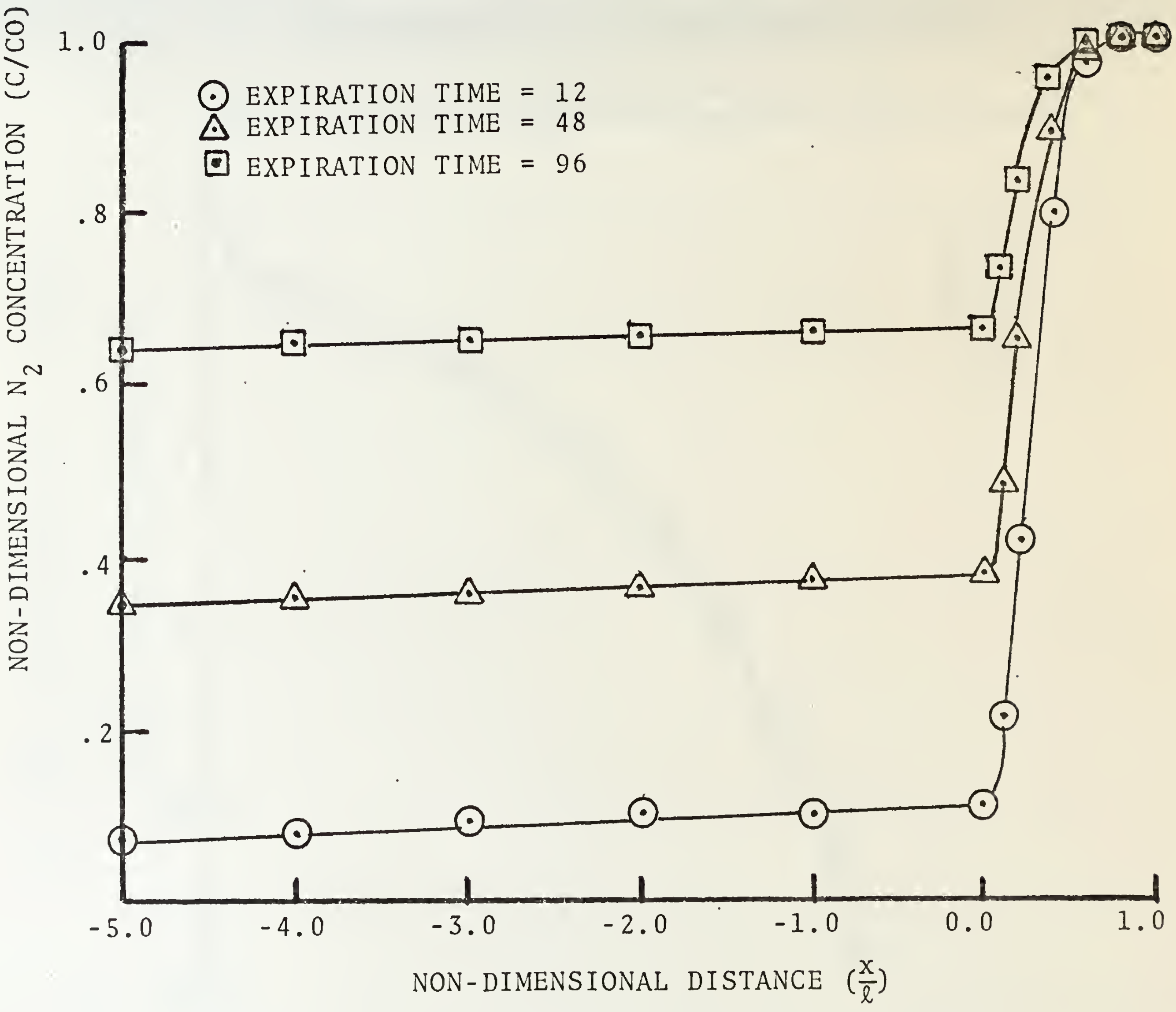

FIGURE 8. N_2 CONCENTRATION DURING EXPIRATION
$N = 10$, $T_{in} = 96$

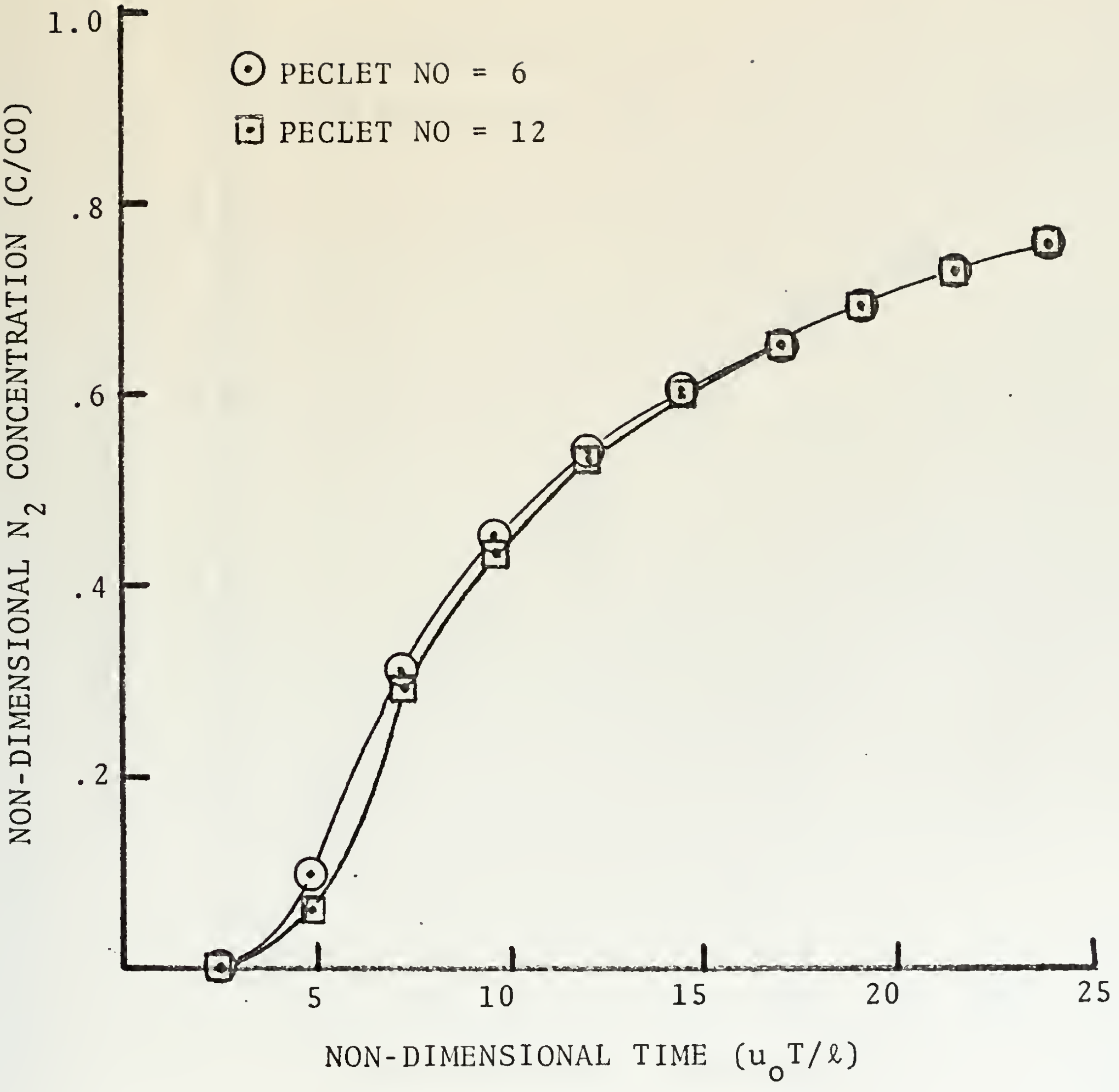

FIGURE 9. N_2 WASHOUT CURVE, T_{in} = 12.

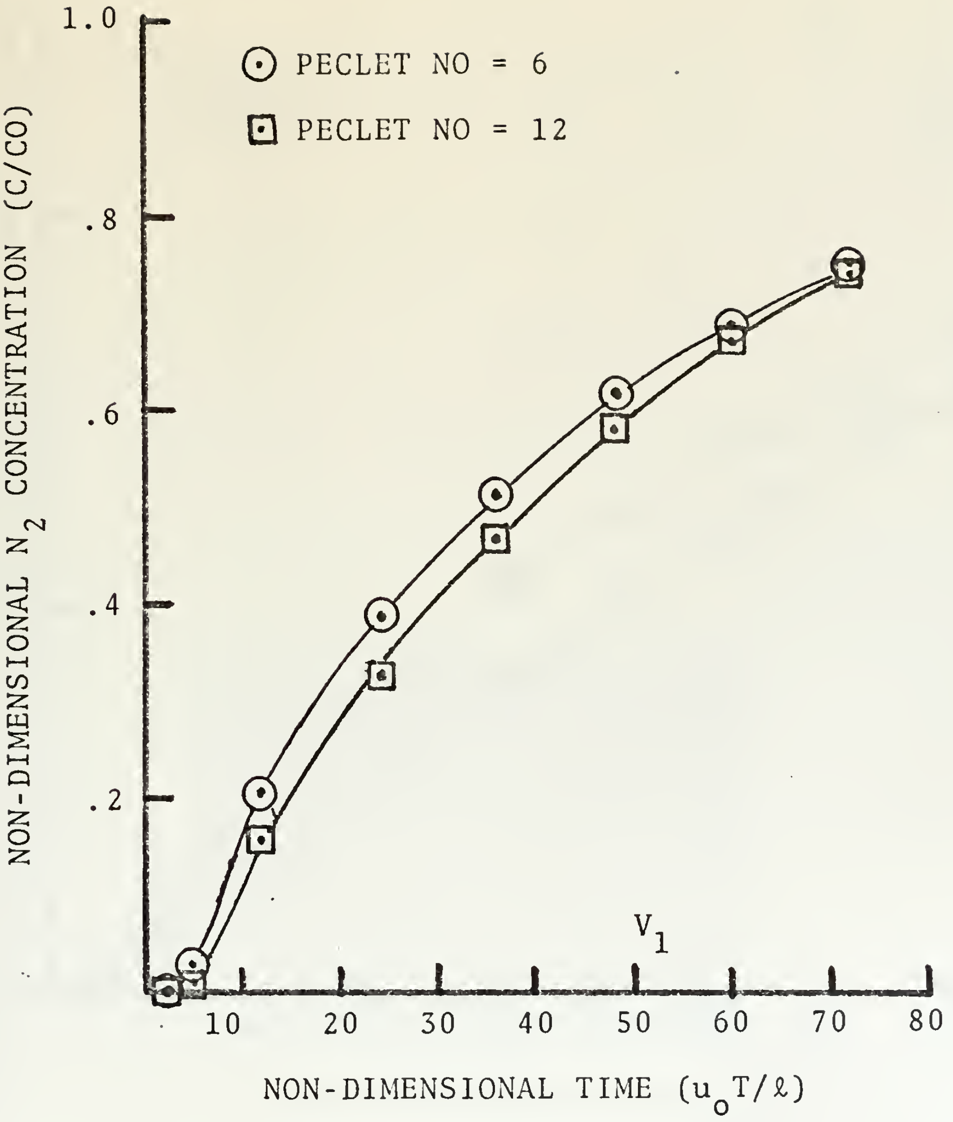

FIGURE 10. N_2 WASHOUT CURVE, T_{in} = 48.

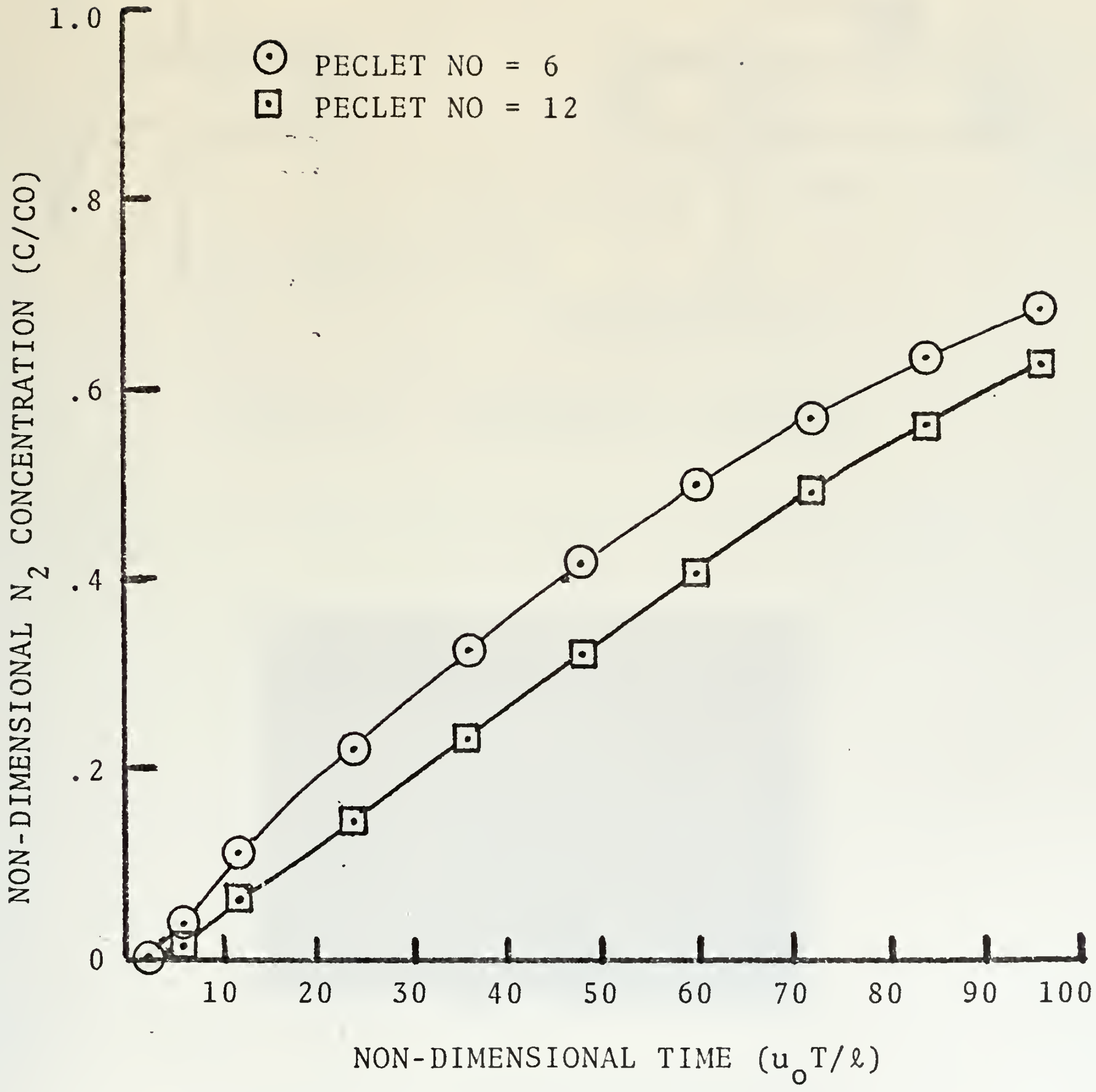

FIGURE 11. N_2 WASHOUT CURVE, T_{in} = 96.

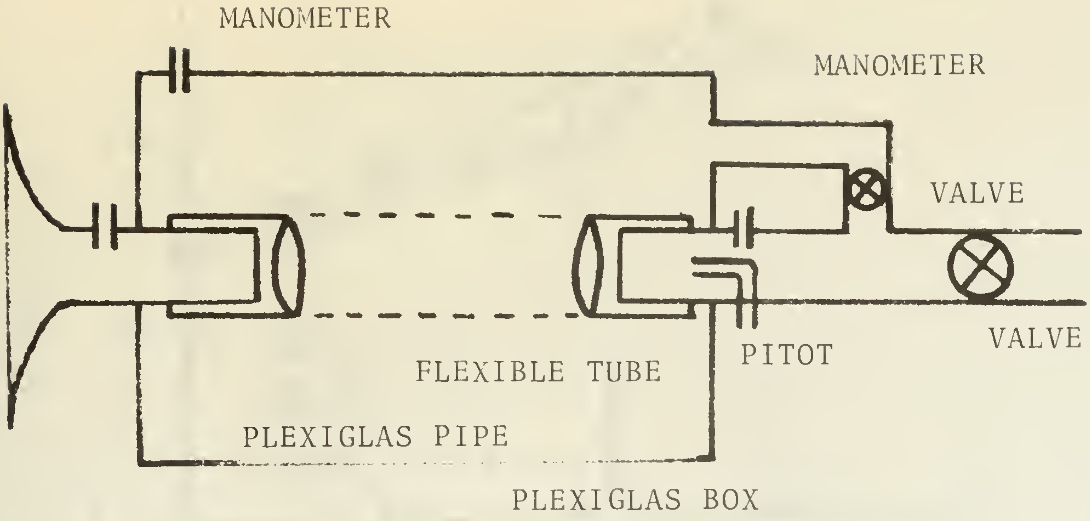

FIGURE 12. EXPERIMENTAL APPARATUS.

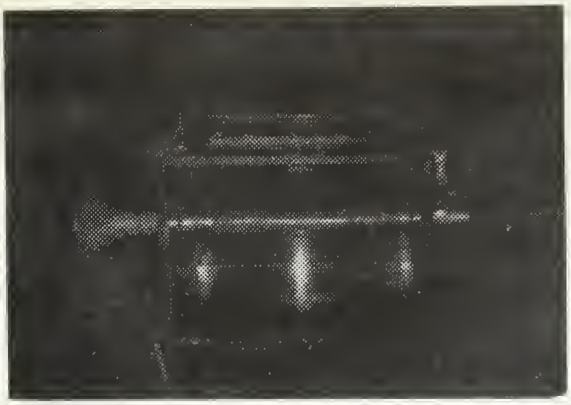

FIGURE 13. EXPERIMENTAL APPARATUS

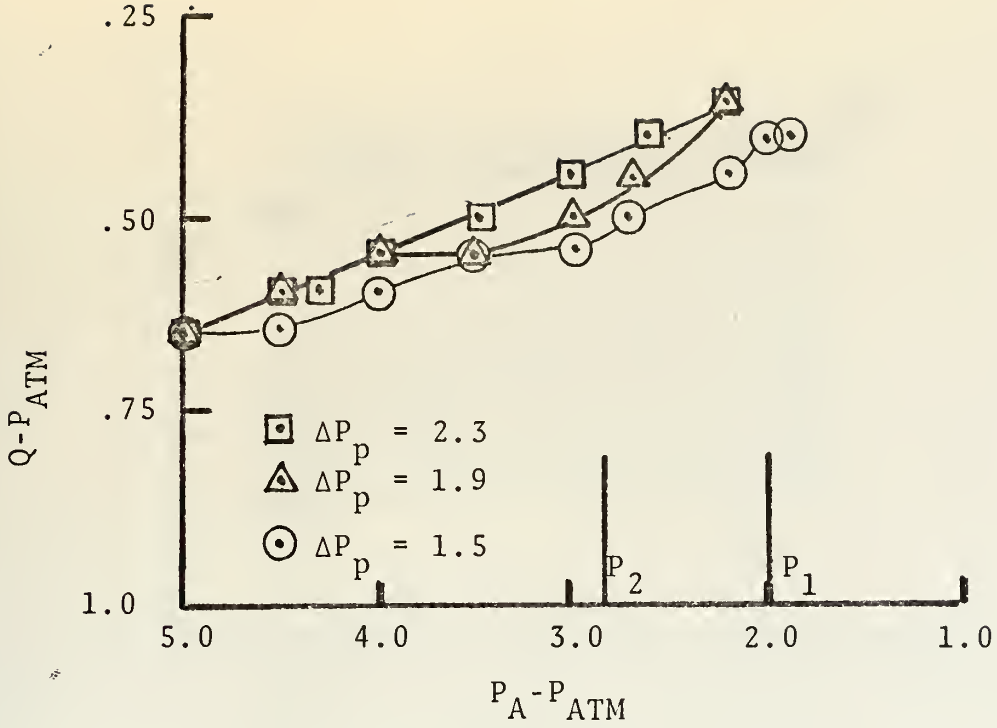

FIGURE 14. FLOW VARIATION WITH ALVEOLAR PRESSURE, 40 = 5.5.

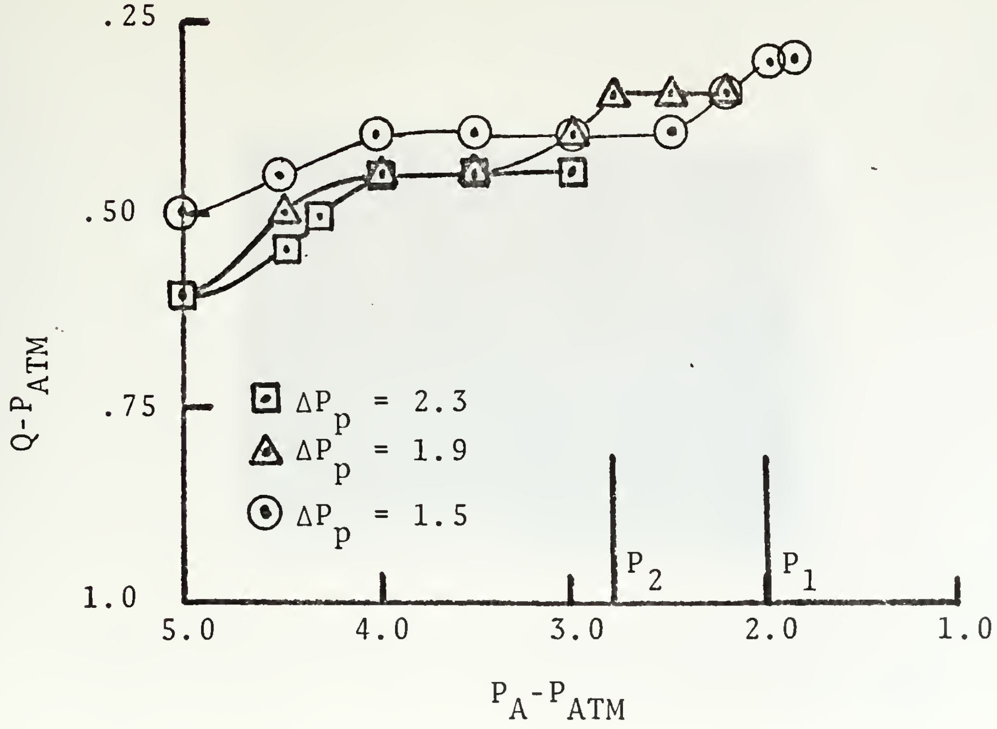

FIGURE 15. FLOW VARIATION WITH ALVEOLAR PRESSURE, 40 = 4.0.

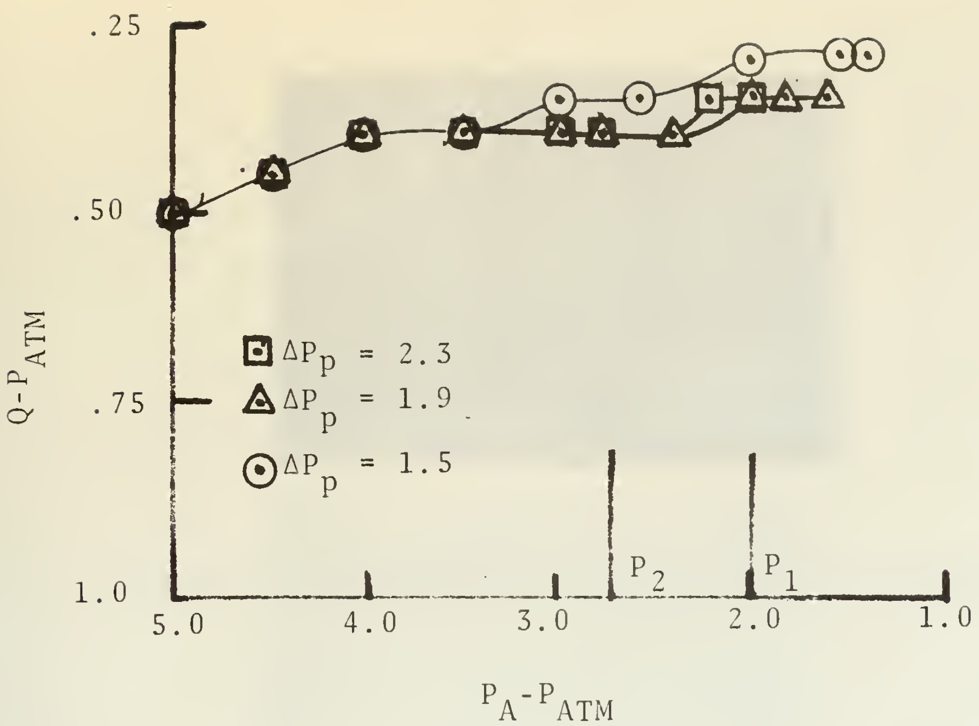

FIGURE 16. FLOW VARIATION WITH ALVEOLAR PRESSURE, 40 =2.5.

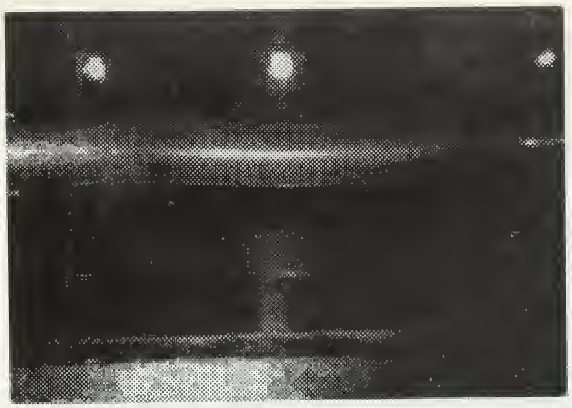

FIGURE 17. "PARALLEL" FLOW IN COLLAPSIBLE TUBE.

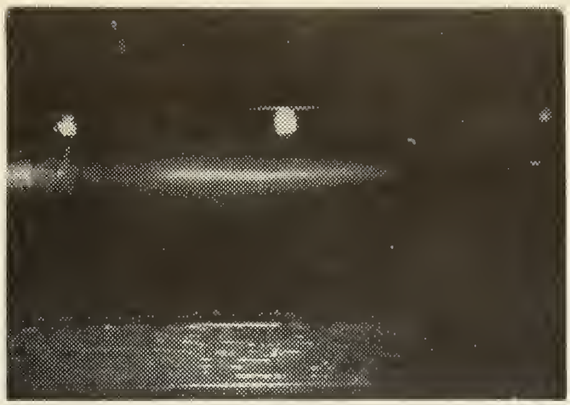

FIGURE 18. "BALLOON" FLOW IN COLLAPSIBLE TUBE.

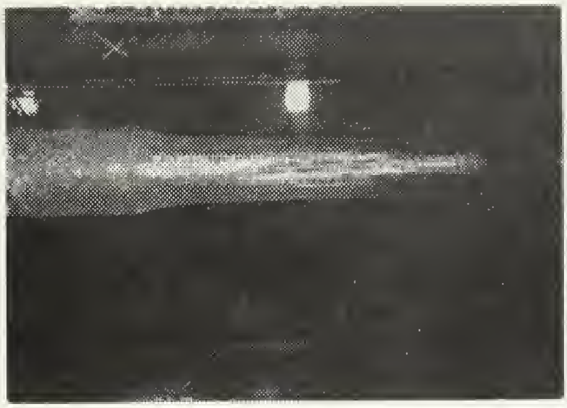

FIGURE 19. STRIATIONS PRIOR TO COLLAPSE.

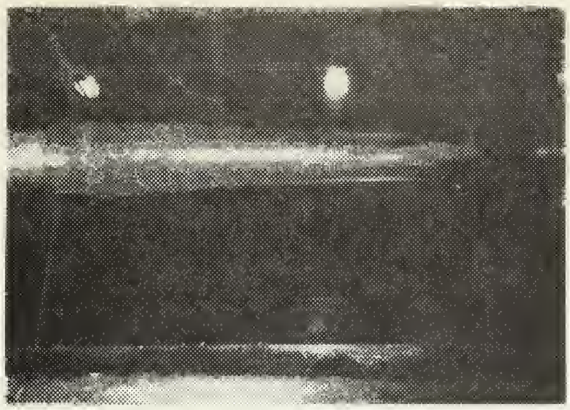

FIGURE 20. UNSTEADY COLLAPSE.

APPENDIX A

NUMERICAL SOLUTION DATA

N_2 Concentration during Inspiration, $T_{in} = 4.8$

Distance (x/ℓ)	Concentration (c/c_0) N = 6	N = 8	N = 10	N = 12
-5.0	.000	.000	.000	.000
-4.0	.000	.000	.000	.000
-3.0	.006	.002	.000	.000
-2.0	.050	.030	.017	.008
-1.4	.130	.103	.087	.062
-1.0	.216	.193	.172	.154
-0.4	.389	.387	.387	.386
0.0	.545	.549	.554	.559
0.2	1.000	1.000	1.000	1.000
0.6	1.000	1.000	1.000	1.000
0.98	1.000	1.000	1.000	1.000

N_2 Concentration during Inspiration, N = 10

Distance	Concentration (c/c_0)			
(x/ℓ)	T_{in} = 4.8	T_{in} = 12	T_{in} = 48	T_{in} = 96
-5.0	.000	.000	.000	.000
-4.0	.000	.000	.000	.000
-3.0	.000	.000	.000	.000
-2.0	0.17	.000	.000	.000
-1.4	.081	.000	.000	.000
-1.0	.172	.000	.000	.000
-0.4	.387	.000	.000	.000
0.0	.554	.000	.000	.000
0.1	.999	.870	.301	.129
0.2	1.000	.998	.641	.321
0.4	1.000	1.000	.970	.746
0.6	1.000	1.000	.999	.966
0.8	1.000	1.000	1.000	.999
0.98	1.000	1.000	1.000	1.000

N_2 Concentration during Expiration, N = 10

Distance	Concentration (c/c_0)					
(x/ℓ)	$T_{in}=12$	$T_{in}=48$		$T_{in}=96$		
	$T_{ex}=12$	$T_{ex}=12$	$T_{ex}=48$	$T_{ex}=12$	$T_{ex}=48$	$T_{ex}=96$
-5.0	.532	.170	.586	.073	.344	.642
-4.0	.559	.185	.593	.080	.351	.646
-3.0	.586	.201	.601	.087	.358	.646
-2.0	.610	.216	.609	.095	.365	.656
-1.0	.632	.231	.616	.102	.373	.661
0.0	.652	.246	.623	.110	.380	.666
0.1	.853	.439	.720	.216	.479	.728
0.2	.984	.719	.859	.418	.650	.828
0.3	.999	.898	.945	.628	.799	.904
0.4	1.000	.973	.982	.800	.894	.949
0.6	1.000	.999	.998	.969	.979	.988
0.8	1.000	1.000	1.000	.999	.998	.998
0.98	1.000	1.000	1.000	1.000	1.000	.999

Washout Data, T_{in} = 12

T_{ex} (u_0t/ℓ)	Concentration (c/c_0) N = 6	Concentration (c/c_0) N = 12
2.4	.000	.000
4.8	.101	.061
7.2	.317	.298
9.6	.453	.438
12.0	.540	.530
14.4	.603	.597
16.8	.651	.650
19.2	.689	.692
21.6	.721	.726
24.0	.748	.755

Washout Data, $T_{in} = 48$

T_{ex} ($u_0 t/\ell$)	Concentration (c/c_0) N = 6	N = 12
2.4	.000	.000
4.8	.030	.013
6.0	.067	.042
12.0	.207	.157
18.0	.304	.247
24.0	.384	.328
30.0	.453	.400
36.0	.513	.466
42.0	.566	.525
48.0	.612	.578
54.0	.653	.626
60.0	.688	.668
66.0	.720	.705
72.0	.748	.738

Washout Data, T_{in} = 96

T_{ex} $(u_0 t/\ell)$	Concentration (c/c_0) N = 6	n = 12
2.4	.000	.000
4.8	.010	.002
6.0	.034	.016
9.8	.086	.046
12.0	.110	.061
24.0	.224	.144
36.0	.325	.231
48.0	.417	.320
60.0	.499	.407
72.0	.571	.488
84.0	.634	.562
96.0	.688	.629
108.0	.735	.687
120.0	.774	.737

APPENDIX B

THIN-WALLED COLLAPSIBLE TUBE DATA

All pressures are inches of water below atmospheric pressure. P_1 is 2.0 for all cases.

$L/D = 5.5,\ \Delta P_p = 2.3$

P_2	P_A	q
2.85	5.0	.65
2.85	4.5	.60
2.85	4.3	.60
2.85	4.0	.55
2.85	3.5	.50
2.85	3.0	.45
2.85	2.8	.45
2.85	2.6	.40

$L/D = 5.5,\ \Delta P_p = 1.9$

2.80	5.0	.65
2.80	4.5	.60
2.80	4.0	.55
2.80	3.9	.55
2.80	3.5	.55
2.80	3.0	.50
2.80	2.6	.45
2.80	2.4	.40
2.80	2.25	.40
2.80	2.2	.35

$L/D = 5.5, \Delta P_p = 1.5$

P_2	P_A	q
2.75	5.0	.65
2.75	4.5	.65
2.75	4.0	.60
2.75	3.5	.55
2.75	3.0	.55
2.75	2.75	.50
2.75	2.2	.45
2.75	2.0	.40
2.75	1.9	.40

$L/D = 4.0, \Delta P_p = 2.3$

2.80	5.0	.60
2.80	4.5	.55
2.80	4.3	.50
2.80	4.0	.45
2.80	3.5	.45
2.80	3.0	.45

$L/D = 4.0,\ \Delta P_p = 1.9$

P_2	P_A	q
2.80	5.0	.60
2.80	4.5	.50
2.80	4.0	.45
2.80	3.9	.45
2.80	3.5	.45
2.80	3.0	.40
2.80	2.8	.35
2.80	2.5	.35
2.80	2.1	.35

$L/D = 4.0,\ \Delta P_p = 1.5$

2.75	5.0	.50
2.75	4.5	.45
2.75	4.3	.40
2.75	4.0	.40
2.75	3.5	.40
2.75	3.0	.40
2.75	2.75	.40
2.75	2.5	.40
2.75	2.2	.35
2.75	2.0	.30
2.75	2.0	.30
2.75	2.1	.30

$L/D = 2.5$, $\Delta P_p = 2.3$

P_2	P_A	q
2.75	5.0	.50
2.75	4.5	.45
2.75	4.3	.40
2.75	4.0	.40
2.75	3.5	.40
2.75	3.0	.40
2.75	2.75	.40
2.75	2.2	.35
2.75	2.0	.35

$L/D = 2.5$, $\Delta P_p = 1.9$

2.70	5.0	.50
2.70	4.5	.45
2.70	4.0	.40
2.70	3.9	.40
2.70	3.5	.40
2.70	3.0	.40
2.70	2.7	.40
2.70	2.4	.40
2.70	2.0	.35
2.70	1.8	.35
2.70	1.6	.35

$L/D = 2.5$, $\Delta P_p = 1.5$

P_2	P_A	q
2.65	5.0	.50
2.65	4.5	.45
2.65	4.0	.40
2.65	3.5	.40
2.65	3.0	.35
2.65	2.6	.35
2.65	2.0	.35
2.65	1.8	.30
2.65	1.5	.30
2.65	1.6	.30

COMPUTER NUMERICAL SOLUTION FOR GAS MIXING IN A SIMPLE MODEL OF THE LUNG

```
      DIMENSION CNW(124),CTN(124)
      NP=10
      WRITE (6,250) NP
  250 FORMAT (5X,'PECLET NO. IS',3X,I3,//)
      T=0.0
      DELX=0.2
      JE=124
      NM=2
      JE1=JE-1
      DO 10 I=1,4000
      X=-5.0
      D=1.0
      F=-1.0
      A=1.0
      DELX=.2
      DELT=(.3*(DELX**2))/D
      IF (I.GT.1) GO TO 111
      WRITE (6,150)
  150 FORMAT (5X,'T EQUALS   0.0',/)
      WRITE (6,300)

      SET THE INITIAL CONDITIONS

      DO 110 K=1,JE
      CTN(K)=1.0
      IF (X.LT.0.) GO TO 30
      DELX=.01
   30 X=X+DELX
      WRITE (6,310) X,CTN(K)
  110 CONTINUE
      X=-5.0
      DELX=.2
  111 CTN(1)=0.0
      T=T+DELT
      MOD=(I/500)*500
      IF (MOD.EQ.I) GO TO 50
      GO TO 70
   50 WRITE (6,200) T
  200 FORMAT (//,5X,'T EQUALS',3X,F8.4,/)
      WRITE (6,300)
  300 FORMAT (5X,'X EQUALS',5X,'C EQUALS',/)
   70 DO 100 J=2,JE1

      CALCULATE CONCENTRATION FROM MOUTH TO ALVEOLI
                     FOR EACH TIME STEP

   11 IF (X.LT.0.) GO TO 40
      DELX=.01
      DELT=(.3*(DELX**2))/D
      A=(1-X)**(-2)
      D=.2+.8*((1-X)**(2*NM))
      FB=(.4+1.6*(1-X)**(2*NM))/(NP*(1-X))-1/A
      F=(NM/NP)*(-1.6)*(1-X)**(2*NM-1)+FB
   40 X=X+DELX
      G=(D/NP)*(CTN(J+1)-2.*CTN(J)+CTN(J-1))/(DELX**2)
      H=(CTN(J+1)-CTN(J-1))/(2.*DELX)
      CNW(J)=CTN(J)+DELT*(G+(F*H))
      IF (MOD.EQ.I) GO TO 55
      GO TO 100
   55 WRITE (6,310) X,CNW(J)
  310 FORMAT (5X,F8.4,3X,F12.7)
  100 CONTINUE
      X=X+DELX

       IMPOSE B.C.  DC/DX=0  AT ALVOELAR END

       CNW(JE)=4./22.*(9*CNW(JE1)-18./4.*CNW(JE-2)+CNW(JE-3))
```

```
      DO 7 L=2,JE
      CTN(L)=CNW(L)
    7 CONTINUE
      IF (MOD.EQ.I) GO TO 60
      GO TO 10
   60 WRITE (6,310) X,CTN(JE)
   10 CONTINUE

      STOP INSPIRATION, START EXPIRATION

      CTN(1)=0.0
      WRITE (6,700)
  700 FORMAT (//,5X,'STOP INSPIRATION, START EXPIRATION',//)
      DO 15 I=1,6000
      DELX=.2
      X=-5.0
       D=1.0
      A=1.0
      F=1.0
      DELT=(.3*(DELX**2))/D
      T=T+DELT
      IF (I.GT.500) GO TO 17
      MAD=(I/20)*20
      IF (MAD.NE.I) GO TO 17
      WRITE (6,200) T
      WRITE (6,300)
      WRITE (6,310) X,CTN(1)
   17 MOD=(I/500)*500
      IF (MOD.EQ.I) GO TO 205
      GO TO 75
  205 WRITE (6,200) T
      WRITE (6,300)
      WRITE (6,310) X,CTN(1)
   75 DO 105 J=2,JE1

      CALCULATE CONCENTRATION FROM MOUTH TO ALVEOLI
                FOR EACH TIME STEP

      IF (X.LT.0.) GO TO 45
      DELX=.01
      DELT=(.3*(DELX**2))/D
      A=(1-X)**(-2)
      D=.2+.8*((1-X)**(2*NM))
      FB=(.4+1.6*(1-X)**(2*NM))/(NP*(1-X))+1/A
      F=(NM/NP)*(-1.6)*(1-X)**(2*NM-1)+FB
   45 X=X+DELX
      G=(D/NP)*(CTN(J+1)-2.*CTN(J)+CTN(J-1))/(DELX**2)
      H=(CTN(J+1)-CTN(J-1))/(2.*DELX)
      CNW(J)=CTN(J)+DELT*(G+(F*H))
      IF (MOD.EQ.I) GO TO 90
      GO TO 105
   90 WRITE (6,310) X,CNW(J)
  105 CONTINUE
      X=X+DELX

      IMPOSE B.C.  DC/DX=0  AT ALVOELAR END

      CNW(JE)=4./22.*(9*CNW(JE1)-18./4.*CNW(JE-2)+CNW(JE-3))

      IMPOSE B.C.  DC/DX=0  AT MOUTH

      CTN(1)=4./22.*(9.*CNW(2)-18./4.*CNW(3)+CNW(4))
      DO 9 L=2,JE
      CTN(L)=CNW(L)
    9 CONTINUE
      IF (MOD.EQ.I) GO TO 65
      GO TO 15
   65 WRITE (6,310) X,CTN(JE)
   15 CONTINUE
      STOP
      END
```

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13. ABSTRACT

The range of fluid dynamics applications in the lung is vast, encompassing many interesting problems rarely touched in any other single engineering area. A survey of recent work in this field was conducted. Additionally, a numerical solution for gas mixing in a simple model of the lung was developed indicating its application to a wide range of similar problems. Finally, new parameters were experimentally investigated in flow through a thin-walled collapsible tube. Results indicated the effects of the parameters to be significant in the flow of blood in the pulmonary capillaries as controlled by the surrounding aveolar pressure.

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